

## Disseminated Tuberculosis Involving Allograft in a Renal Transplant Recipient

Sir,

Tuberculosis (TB) is an important opportunistic infection encountered in postrenal transplant patients in developing countries. The frequency of *Mycobacterium tuberculosis* infection in most developing countries ranges from 1.2% to 6.4% and upto 12%.<sup>[1]</sup> In countries where TB is endemic, like India, the incidence in patients on maintenance dialysis is 8.7%, and that in renal allograft recipients is 12.3%.<sup>[2]</sup> Here, we present a renal transplant recipient presenting with multiple abscesses in the renal allograft, brain, retroperitoneum, and seminal vesicles of tuberculous origin.

A 44-year-old nonhypertensive post renal transplant male patient presented with complaints of fever, malaise, and headache after 2 years of transplantation. He had no history of TB in the past. The patient was on two drug immunosuppression (tacrolimus and prednisolone) his baseline serum creatinine was 1.35 mg/dl. No palpable lymph nodes were present. On ultrasonography (USG), few hypoechoic lesions were noted at the lower pole of renal allograft. Later on contrast enhanced computed tomography (CECT) of Abdomen was performed, revealed multiple, confluent peripherally enhancing hypodense lesions in the renal allograft, retroperitoneum, in the left seminal vesicle, multiple mesenteric lymphadenopathy with central necrosis [Figure 1]. CT thorax revealed no any abnormality. Hence, the patient was kept on 4-drug antituberculous therapy (isoniazid, levofloxacin, ethambutol, and pyrazinamide). During the course of treatment, the patient developed convulsion. Hence CECT brain was performed revealed abscesses in the

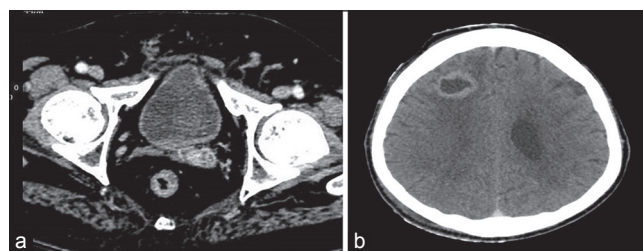


**Figure 1:** Coronal image of arterial phase of computed tomography scan of abdomen showing two peripherally enhancing lesion at lower pole of renal allograft

right anterior parietal lobe and in the right cerebellum [Figure 2]. Hence, drug rifampicin was added. The patient improved and developed liver cirrhosis and graft dysfunction due to the toxicity of drugs.

The risk of developing TB after kidney transplantation has been estimated to be 50-100 times higher than in the general population. A global review on TB estimated the median time for onset at 9 months post transplantation.<sup>[3]</sup> Renal allograft recipients present with extra-pulmonary TB (51.8%). Among them, disseminated TB is the most common (19.3%), followed by pyrexia of unknown origin (15.7%), TB lymphadenopathy (4.8%), involvement of skin and soft tissue (4.2%), intestine (3%), central nervous system (1.8%), bone (1.2%), pericardium (1.2%), or urinary tract (0.6%).<sup>[4]</sup> TB in renal transplant recipients develops mainly because of the reactivation of an old, preexisting focus. Disseminated nature of TB in transplant recipients might be the result of the delay in diagnosis due to an atypical presentation or the result of excessive immune-suppression. The classical risk factors are a longer period of hemodialysis, pretransplant diabetes mellitus, induction immunosuppression with anti-CD25 monoclonal antibodies, >3 episodes of rejection, treatment of rejection with antilymphocyte globulin, bolus corticosteroids, chronic liver disease, deep mycoses, pneumocystis pneumonia and nocardia infections. For diagnosis of renal allograft TB, all modalities can be utilized; however, due to its superficial location, USG obviously plays a major role. Multidetector CT can easily identify calcification, renal scars, mass lesions, urothelial thickening, uneven caliectasis and lack of pelvic dilatation. In our patient, there was no evidence of active pulmonary TB or any focus of infection before transplantation and the donor was also screened negative in urine and systemic culture for acid-fast *Bacilli*. Hence, the encountered infection was proved to develop de novo due to the immunosuppressed status.

The presentation of the disseminated TB differs in solid organ recipients, and a high index of suspicion is



**Figure 2:** (a) Axial image of the venous phase of computed tomography scan showing peripherally enhancing lesions in the left seminal vesicle. b) Axial image of arterial phase of computed tomography scan of brain showing peripherally enhancing lesion in the right parietal lobe of cerebrum

important in diagnosing the problem. Diagnosis is along conventional lines though the sensitivity and specificity of the investigations vary. Tailored immunosuppression, with stringent monitoring of the drug levels, is expected to decrease the incidence and prevalence in the future. The duration of treatment has to be prolonged, and secondary prophylaxis has to be considered. Drug resistance and atypical mycobacterial infections are emerging problems and should be suspected in the nonresponding patients.

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### Conflicts of interest

There are no conflicts of interest.

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